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HAND
CLINICS

Etiology and pathology

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Etiology

Trauma

The etiology of neurogenic thoracic outlet syndrome (TOS) is believed to be a combination of neck trauma plus an anatomic predisposition. The basis for regarding neck trauma as the primary etiology stems from observations in most of a few thousand patients whose symptoms of pain in their necks and arms and paresthesia in their hands developed shortly after a motor vehicle accident. This observation was followed by microscopic studies that demonstrated significant histologic changes in the scalene muscles of patients with neurogenic TOS [1]. Neck trauma may occur as a single acute incident or it can develop insidiously following repeated small episodes of neck strain.

Acute neck trauma

Motor vehicle accidents have a high incidence of whiplash injuries. This is the most common mechanism causing neurogenic TOS. More than half of the patients the authors see with neurogenic TOS developed their symptoms following a motor vehicle accident [2]. Other investigators also have noted a high incidence of neck trauma [3]. Studies of whiplash injuries following motor vehicle accidents by several authors revealed that more than 30% of these patients developed symptoms of neurogenic TOS; although most patients responded to conservative therapy, a fair number went on to surgical decompression of the thoracic outlet [4–6].

Repetitive stress and posture

The next most common cause of neurogenic TOS is repetitive stress injury in the work place. This develops in people who work at keyboards, on telephones, on assembly lines, or who sit in one position for several hours at a time. The specific cause is repeated small traumata to the neck muscles over a prolonged period of time either by frequently turning the neck back and forth, holding a telephone by bending the ear against the shoulder, or poor posture causing muscle imbalance. In muscle imbalance, some muscles are overused and others underused [7]. TOS attributable to these causes frequently is associated with cumulative trauma syndrome, which includes carpal tunnel, pronator tunnel, radial tunnel, and cubital tunnel syndromes together with TOS.

Support for the influence of posture on TOS symptoms comes from a survey of more than 1000 dentists, 98% of whom responded [8]. In this group of dental practitioners, there was a 29% incidence of pain and paresthesia noted in the upper extremities. In a similar study of dental hygienists, 26% were noted to have symptoms of carpal tunnel syndrome. This emphasizes the importance of posture and position in the etiology and treatment of neuropathies caused by muscle imbalance or overuse [8].

Other etiologies that may play a small role in causing neurogenic TOS are large, heavy breasts

Chapter on Etiology and Pathology: Fig. 1: Variations in scalene triangle–Fig. 83-3, Pge 1187 Fig. 2: Scalene muscle histology–Fig. 83-2, Pge 1186

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and carrying heavy backpacks. These causes are uncommon but do occur.

Anatomic predispositions

Anatomic variations and anomalies probably play a secondary role in etiology. Congenital bands and ligaments are observed in a large majority of patients with neurogenic TOS and several different types have been recognized and categorized [9]. These anatomic variations, however, have been observed in more than half of anatomic dissections in cadavers [10]. These bands and ligaments have been present since birth. In most patients with TOS, they only became associated with symptoms following neck injury. The authors therefore regard these anatomic findings as predisposing factors rather than the causative agent.

Cervical ribs also are regarded as predisposing factors in most patients in whom they are seen. As with anatomic variations and bands, cervical ribs have been present since birth. In 80% of patients with cervical ribs, symptoms did not develop until after a neck injury [11]. One exception has been observed in patients with complete cervical ribs. In this group the abnormal rib, as opposed to neck trauma, may be the primary etiology of the symptoms, as only 50% of patients developed symptoms following neck trauma.

Anatomic variations in the scalene triangle may play a significant role in predisposing patients to develop TOS symptoms. Anatomic observations of these variations were made in cadavers and compared with similar observations on patients with TOS during surgery [12]. The nerve roots of the brachial plexus emerged from the apex of the scalene triangle in 40% of cadaver control subjects compared with 83% of patients with TOS. Interdigitating muscle fibers between the anterior and middle scalene muscles, fibers that put stress on the nerve roots, were present in 40% of cadavers and 75% of patients with TOS. Adherence of nerves to the anterior scalene muscle occurred in 29% of cadavers compared with 90% of patients with TOS, and adherence of nerves to the middle scalene muscle was seen in 32% of cadavers compared with 61% of patients with TOS. Another observation is that some scalene triangles are narrower than others; patients with TOS tend to have narrower triangles than the average seen in cadavers (Fig. 1) [12].

Pathology

If the site of pathology in neurogenic TOS is the scalene muscles, are there abnormalities in these muscles to support this hypothesis? In 1935, the first report to discuss muscle pathology appeared [13]. It described hypertrophy, degeneration, and fibrosis in the anterior scalene muscle. These studies used formalin fixation, technology that is now known to alter many delicate features of muscle.

Only two histologic studies of scalene muscles have been published to date using modern techniques of rapid freezing with liquid nitrogen, deep freezer storage, and enzymatic tissue stains [1,14]. This permits detection of subtle changes in muscular cytoarchitecture. In both of these studies a nonspecific finding of Type I fiber predominance associated with Type II fiber atrophy and pleomorphism was observed. In addition, one of the studies noted a significant increase in connective tissue from an average of 14.5% in control subjects to 37.8% in patients with TOS, which represents muscle scarring (Fig. 2) [1].

Pathophysiology

Based on the observations noted previously, namely the history of neck trauma, variations in normal anatomy, and histologic changes in scalene muscles, it is possible to propose a reasonable scenario to explain the pathophysiology of neurogenic TOS. The underlying pathology in most patients is scarring of the scalene muscles caused by hyperextension neck injuries. The tight scalene muscles cause neck pain and occipital headaches, which usually develop within a few days of the accident. As scarring in the muscles develops, the muscles compress the brachial plexus, eliciting the symptoms of pain, paresthesia, and weakness of the upper extremity. The onset of extremity symptoms may be delayed a few days to weeks, and in a few patients, even months, as it takes time for scar tissue to develop and compress. In many patients the extremity symptoms thus may be regarded as the result of the healing process of the muscles.

Summary

Until the 1920s, TOS was believed to be a vascular condition caused by compression of the subclavian artery by a congenital anomaly, either a cervical rib or tight anterior scalene

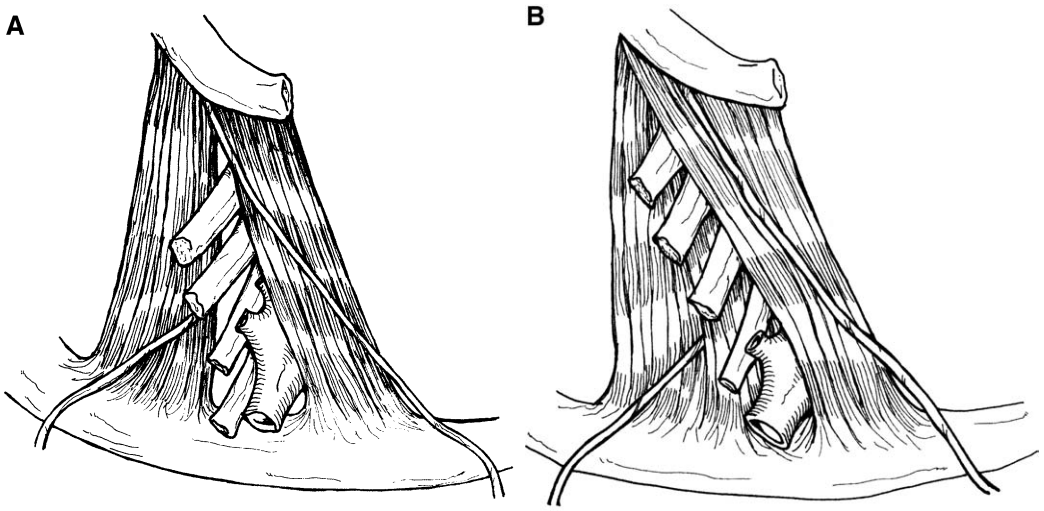

Fig. 1. Variations in the scalene triangle. (*A*) The usual relations found in most cadavers. The triangle is wider and the nerves emerge lower in the triangle than in most patients with TOS. (*B*) A narrow triangle in which the nerves emerge high and are touching the muscles as they emerge. *Reproduced from* Sanders RJ, Roos DB. The surgical anatomy of the scalene triangle. Contemp Surg 1989;35:11–6; with permission.

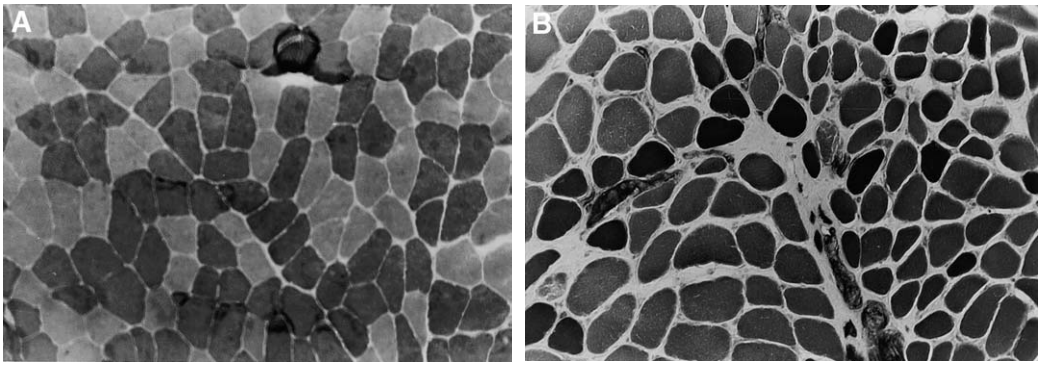

Fig. 2. Scalene muscle histology. (*A*) Control subject. (*B*) Patient with TOS. Note the increase in connective tissue around each muscle fiber in the patient with TOS. Note too the equal distribution of light and dark staining fibers (Type 1 and Type 2 fibers, respectively) in the control subject compared with the reduction in number and pleomorphism of the dark staining Type 2 fibers. *Reproduced from* Sanders RJ, Jackson CGR, Banchero J, Pearce WH. Scalene muscle abnormalities in traumatic thoracic outlet syndrome. Am J Surg 1990;159:231–6; with permission.

muscle. Today it is regarded primarily as a neurologic condition caused by neck trauma injuring and scarring the scalene muscles.

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